ing is continued outward and downward along the attachment of the diaphragm for a distance of eight inches. This flap is then turned outward and downward, leaving a triangular opening with the diaphragm edge presenting. It gives ready access to both sides of the diaphragm and at the same time perfect exposure to the structures, such as the splenic pedicle and vessels which may be injured in either of the other approaches.

Separation of adherent viscera and ligation of all bleeding points, and replacement of viscera, can be accomplished with certainty. Closure of the defect is simple and is usually accomplished with two or three rows of a forty-day chromic gut.

After the diaphragm has been repaired the costochondral flap is replaced and the cut edge of the diaphragm is included in the running chromic stitch which closes the lateral prolongation of the incision.

The paramedian incision is then closed in the same manner, completing the operation. Even in difficult cases the time of operation should not take more than thirty minutes. No subsequent trouble has been encountered with the fracture of the costochondral flap. No special instruments are necessary.

In the presence of adherent viscera it furnishes the ideal approach.

Many cases are not surgical. Surgery may be contraindicated by age, physical condition, or personal preference. In these, great relief may sometimes be obtained by increasing the intrathoracic pressure. These patients may develop a certain emphysema by blowing rubber balloons two or three times a day. The improvement, following this simple procedure, has been startling in some cases.

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EMILE HOLMAN, M. D. (Stanford University Hospital, San Francisco).—Doctor Schiffbauer has very ably and very completely covered the subject of diaphragmatic hernia due to trauma. Physicians generally, and industrial and traumatic surgeons in particular, will do well to bear in mind the possibility of such a hernia incident to any severe trauma to abdomen or to chest. If a diagnosis of hernia through the diaphragm is made, and if abdominal symptoms predominate, the abdominal approach is indicated, preferably through a long incision parallel to the costal margin extending through the linea alba medially and to the anterior axillary line laterally. An excellent exposure of the diaphragm is obtained, and after reduction of the hernial mass it is sometimes possible to identify and crush the phrenic nerve through the rent, if this is deemed necessary to approximate the edges of the rent.

Occasionally, in the abdominal approach, it is difficult to withdraw the abdominal viscera through the rent because of the negative pressure in the thoracic cavity. This can be simply overcome by the passage of a small catheter through the rent alongside the herniated mass, and by the introduction through it of air into the thoracic cavity above the diaphragm, thus equalizing the pressures in the two cavities.

VITAMIN-DEFICIENCY STATES: THEIR RECOGNITION AND TREATMENT*

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PART I

THE concept that disease may be due to deficiency of vitamins has received such widespread proof, and has been so universally accepted, that our clinical interest must now turn to means of recognizing and treating vitamin-deficiency states as well as to a study of the events leading to development of them. The importance of establishing these points lies principally in the fact that vitamin-deficiency states are an unusual group of diseases in that they can be entirely prevented and that, with the exception of a few extreme instances, they are completely amenable to cure.

* Read before the Pediatric Section of the California Medical Association at the sixty-seventh annual session, Pasadena, May 9–12, 1938.

CHARACTER OF DEFICIENCY STATES

For purposes of a clearer understanding of deficiency diseases and the difficulties surrounding recognition of them, such a simple grouping as that presented in Table 1 is of considerable value. In the first group are those patients who, with a marked degree of deficiency, present a classical deficiency disease, as xerophthalmia, beriberi, or pellagra. Such a condition depends on definite pathologic changes in the tissues, it can be recognized by clinical means without recourse to special tests, and it is infrequently encountered in this country.

In a second group may be placed those diseases which are usually due to a moderate degree of deficiency, such as the multiple neuritis of chronic alcoholism, nutritional night blindness, certain types of macrocytic anemia and glossitis. In these cases the deficiency is less marked than in the cases of the preceding group, the symptoms depend on physiologic and pathologic changes, and the diagnosis may rely on the use of special diagnostic tests.

In many respects the sequence of events in these two groups of "deficiency states" may be likened to those occurring in patients with diabetes, nephritis or cardiac disease. Patients with a marked degree of vitamin deficiency may be said to be analogous to diabetic or nephritic patients who are in coma, or to cardiac patients who are decompensated and in whom the pathologic changes are extensive and the diagnosis may be made by clinical means alone. Patients with less marked degrees of deficiency, in whom the pathologic changes are less obvious but the physiologic ones very definite, may be considered analogous to patients with definite diabetes, nephritis or heart disease, in whom the symptoms may depend as much on physiologic as on pathologic changes, and in which special tests may or may not be required before a diagnosis can be established. The frequency with which deficiency disease of moderate degree occurs has not been clearly established, but these conditions are apparently common enough to be encountered by most physicians at fairly regular intervals, and to constitute at present the usual case of vitamin deficiency disease.

In a third group may be placed those conditions in which the deficiency is mild and in which recognition of the deficiency may be said principally to depend upon physiologic or chemical alterations rather than on pathologic ones. In fact, recognition of this group of cases in which clinical changes are often absent has depended on the development of special tests, such as the biophotometric determination of dark adaption of the eyes in vitamin A deficiency and the chemical measurement of the saturation of the tissues with vitamin C. The clinical significance of this group of "deficiency states" has not been settled. To carry further the previously mentioned analogy, one might say that this group corresponds to those diabetics in which the diagnosis can be made only after careful studies of the tolerance to dextrose, or with those nephritics who do not present clinical signs of renal

TABLE 1.—*The Character of Vitamin-Deficiency States*

Degree of Deficiency	Clinical Signs	Essential Changes	Incidence	Diagnosis
Marked	Classical Deficiency Disease	Extensive Pathologic	Rare	Clinical findings
Moderate	Variable	Pathologic and Physiologic	Occasional	Clinical findings Laboratory tests
Mild	Absent? Preclinical?	Physiologic	Common	Laboratory tests

disease and in whom the diagnosis depends on careful study of the urinary sediment or of the ability to concentrate urine.

It seems probable that deficiency states falling into the third group will prove to be quite common. However, until much more evidence has accumulated in regard to the physiologic activity of the vitamins, until there are clear-cut methods of determining the chemical concentration of them in tissues and body fluids, and until the nutritional significance of slightly abnormal values are known, it will be impossible to state, in the case of a single individual, the clinical significance of such a slight deficiency.

METHODS OF PRODUCTION OF DEFICIENCY DISEASES: TYPES OF PATIENTS

It is commonly believed that vitamin-deficiency diseases occur only when the diet is inadequate. While this is probably the most common cause of deficiency disease, there may be other factors of etiologic importance, such as an increased demand for vitamins during pregnancy, during fever and when the basal metabolism is increased, such as loss of or destruction of vitamins after ingestion, and such as abnormalities in gastro-intestinal function. (Table 2.) During pregnancy and lactation, for example, the need for certain vitamins may be increased from 10 to 100 per cent, or even more above the requirement for the normal person.

Vitamin-deficiency states should be looked for particularly among those patients who have been on an inadequate diet in an attempt to reduce weight, or due to alcoholism, economic difficulties, faddism, or to poor therapeutic dietary advice, such as long-continued elimination diets, diets for gastro-intestinal diseases, allergic conditions, and so forth. (Table 3.)

THE DIAGNOSIS OF DEFICIENCY STATES

In considering the diagnosis of deficiency disease it is important to keep in mind the fact that the clinical features of a deficiency disease may vary within wide limits. This is due to the fact that there is great individual variation in the response of patients and also experimental animals to the same deficiency diet, and that it is questionable if a vitamin-deficiency state ever exists in pure form. There is almost always deficiency of more than one of the vitamins in a single patient. In fact McCollum and Simmonds pointed out some time ago that it is questionable if a pure vitamin-deficiency state, with the possible exception of scurvy, ever has been produced in the experimental animal.

The diagnosis of vitamin-deficiency states depends principally on clinical observations, although there is rapidly accumulating laboratory evidence to suggest that certain chemical or biologic tests may be useful in substantiating the diagnosis, or in establishing it in the absence of clear-cut clinical findings. A therapeutic test also may be of considerable value as diagnostic procedure, for now that some of the vitamins may be obtained in crystalline form, the specific response to use of a pure form of a vitamin may be considered significant in establishing a diagnosis in some cases.

VITAMIN A DEFICIENCY

Symptoms.

The most readily appreciated clinical symptoms of vitamin A deficiency have to do with changes occurring in the eyes. These symptoms consist principally of night blindness or inability to see clearly in dusky light, and of irritation of or lesions of the conjunctivæ. In its most exaggerated form, xerophthalmia (which is rarely observed in this country), there may be dryness of the conjunctival tissues, conjunctivitis, softening of the cornea, infection of the eyeball, and blindness.

Night blindness, which occasionally may be due to intra-ocular disease such as retinitis pigmentosa, is usually manifested by difficulty or inability of the patient to adapt his vision to faint illumination, although during the daytime or in bright light he may see perfectly. In questioning a patient in re-

TABLE 2.—*Vitamin, Protein and Mineral Deficiency States May Be Produced as a Result of:*

1. Inadequate intake of essential substances in the diet.
2. Increased requirements during periods of rapid growth, pregnancy, infections, fever, elevated basal metabolic rate.
3. Impaired or altered gastrointestinal function.
4. Altered metabolism of the vitamin.

TABLE 3.—*Suspect and Look for Deficiency States*

When the diet is inadequate because of:
1. Economic difficulty
2. Eccentricities, faddism, ignorance, alcoholism
3. Anorexia, dyspepsia, dysphagia, pain
4. Therapeutic dietary inadequacy

In association with or during
1. Gastro-intestinal diseases
2. Pregnancy, Infection, Hyperthyroidism

TABLE 4.—*Vitamin A Deficiency*

Degree of Deficiency	Clinical Signs	Incidence	Tests
Marked	Xerophthalmia	Very rare	
Moderate	Night blindness Skin lesions Conjunctival lesions Epithelial lesions of mucous surfaces	Rare	1. Biophotometer 2. Scrappings of mucous surfaces
Mild (Preclinical?)	Absent	25 to 75 per cent of all children and adults?	3. Therapeutic

gard to night blindness, particular attention should be paid to vision while driving a car at night, while in a theater or the movies, while walking at night or in the dark, or on entering a house from the bright sunlight. Such patients are apt to bump into things while walking at night, to have difficulty in driving a car at night because they cannot see the road or are dazzled by the lights of an approaching car, or to have difficulty in distinguishing facial features, the hands of a clock, or details of a picture, on entering a dimly lighted room.

A history of conjunctival irritation or dryness, which cannot be explained otherwise, may be obtained in some patients with vitamin A deficiency. Examination of the conjunctivae may reveal pigmentation, or there may be dryness or a granular appearance to the conjunctiva if the lids are held away from the eyeball for a few moments.

There has been discussion of the relation of vitamin A deficiency to cutaneous lesions, to lesions of the genital or urinary tract, and to states of lowered resistance to infections. Since deficiency of vitamin A characteristically leads to thickening or keratinization of epithelial tissues, followed frequently by secondary infection, it might be anticipated that cutaneous lesions would occur in vitamin A deficiency. The characteristic change, which is considered by some authorities to be the first manifestation of deficiency, consists of keratinization of a hard, dry papular type that resembles goose flesh and is most marked on the exterior surfaces of the forearms, legs, and thighs. A somewhat similar keratinization of the mucous membrane of the vagina, leading to a lesion resembling that of atrophic or senile vaginitis, has been reported in vitamin A deficiency. The relation of vitamin A deficiency to renal lithiasis in man is still indefinite, and while there is evidence to substantiate the view that in some cases renal lithiasis may be related to deficiency of vitamin A, in most instances of stone this does not seem to be the case.

The only recognized anti-infective influence vitamin A possesses is in maintaining normal epithelium, which will act as a barrier to infection; and since, so far as is known, the vitamin does not have any influence on immunological processes, it seems obvious that vitamin A in reality is not an anti-infective vitamin.

Diagnosis.

Principal interest at present in the diagnosis of vitamin A deficiency is in the detection of states of mild or partial deficiency in which the clinical diagnosis is not easy (Table 4). When the physician is confronted by a patient whom he suspects may have vitamin A deficiency, he should inquire into the presence of night blindness; and on examining the patient he should look for dryness or pigmentation of the conjunctivae and for a papular cutaneous eruption involving particularly the limbs, especially the exterior surfaces.

If vitamin A deficiency is suspected, there are three methods of diagnosis which may be employed in an attempt to confirm the clinical history suggesting that a deficiency exists or to suggest its presence in the absence of clinical findings. There are (1) a study of the sensitivity to light following partial adaptation to darkness, as determined by biophotometric methods of Jeans and Zentmire; (2) scrapping of the conjunctival, nasal or vaginal mucous membrane, making smears and examining them for keratinized epithelial cells; and (3) administration of vitamin A in crystalline or concentrate form. These methods are all relatively indirect, but if either of the first two tests proves to be positive, and if there is disappearance of these signs or of previously noted night blindness or cutaneous or conjunctival symptoms following administration of vitamin A or carotene, the diagnosis of vitamin A deficiency seems reasonably well established.

Physical and chemical methods for determining the vitamin A content of body fluids and tissues have been developed, but have not as yet proved satisfactory for clinical detection of vitamin A deficiency.

The diagnosis of very mild or "preclinical" deficiency of vitamin A in which clinical signs are absent is made at present only by use of the biophotometer. Patients who have no clinical evidence of vitamin A deficiency, but who have defective adaptation to darkness as revealed by this test, and who have normal adaptation after administration of vitamin A, are considered by some observers to have mild deficiency of vitamin A. The frequency with which "clinic populations" have a positive test with this method is in the neighborhood of from 50 to 75 per cent of all those tested, but the clinical significance of this observation has not been established.

Treatment.

Deficiency of vitamin A usually may be prevented by the use of adequate amounts of the following foods rich in vitamin A or carotene: butter, cream, eggs, cod-liver oil, carrots, and spinach. Fish-liver oils which are particularly rich in vitamin A are those of the halibut, cod, burbot, and tuna.

The daily optimal requirement of vitamin A is not known, but it is probably in the neighborhood of 6,000 to 10,000 U. S. P. units. In the treatment of vitamin A deficiency states, the following measures are of value: (1) a diet containing many foods rich in vitamin A; (2) supplements to the diet of

TABLE 5.—*Thiamin Deficiency (Vitamin B_1)*

Degree of Deficiency	Clinical Signs	Incidence	Tests
Marked	Beriberi	Rare	
Moderate	Peripheral neuritis Cardiovascular disturbances Edema Gastro-intestinal dysfunction?* Anemia?*	Infrequent	1. Calculations Cowgills Formula 2. Therapeutic 3. Amounts of Thiamin in blood and urine
Mild (Preclinical?)	Uncertain mild degree of above	Unknown	

* Changes in these organs are very frequently associated with thiamin deficiency, but are probably due to deficiency of components of the vitamin B complex other than thiamin.

vitamin A in cod or halibut-liver oil in amounts of 10,000 to 25,000 U. S. P. units daily (Jeghers suggests 70,000 U. S. P. units orally for two weeks, and the 25,000 units until the patient is normal); (3) in rare instances by intramuscular injection of cod-liver oil or other substances rich in vitamin A.

VITAMIN B_1 (THIAMIN) DEFICIENCY

Symptoms.

Thiamin is a catylist essential for the oxidation of carbohydrates, and when it is not present in adequate amounts in man the characteristic symptims of peripheral neuritis, circulatory failure, and beriberi occur. (Table 5.)

As Strauss and others have indicated, the diagnosis of alcoholic, diabetic, biliary, gastrogenic and postinfective polyneuritis, polyneuritis of pregnancy, and of Korsakoff's syndrome, have all concealed the true diagnosis of thiamin (vitamin B_1) deficiency. While symptoms of these conditions may be sudden in onset they are generally insidious, and the earliest manifestations usually are heaviness in the legs and tenderness of the calf muscles when they are squeezed. Weakness of the limbs, burning of the soles, and numbness of the dorsum and lower part of the ankles, are next to appear, followed by hypesthesia, which advances up the leg and thigh, and by atrophy of the muscles and of the skin. With these symptomatic changes there usually are associated diminished or absent reflexes in the involved extremities and various degrees of muscle weakness.

The cardiovascular manifestations of thiamin deficiency have been extensively studied by Weiss and Wilkins, and consist principally of dyspnea and palpitation on exertion, tachycardia, and edema. In cases in which the deficiency is greater the clinical picture may be that of cardiac failure with an enlarged area of cardiac dullness or an enlarged shadow on the roentgenogram, signs of right or left heart failure, and extensive edema or anasarca with or without signs of peripheral neuritis. It is obvious that in the absence of marked changes these manifestations do not, for the present at least, comprise a rigid and easily recognized clinical syndrome.

Other manifestations of thiamin deficiency which have been reported over a period of years include gastro-intestinal changes such as anorexia, glossitis, achlorhydria and diarrhea, and changes in the blood such as those indicating anemia. As Strauss has pointed out, there is considerable evidence to suggest that these phenomena are, at least in part if not entirely, manifestations of a deficiency of some portion of the vitamin B complex other than thiamin.

Diagnosis.

The diagnosis of well-established thiamin deficiency is simple if the characteristic triad of symptoms of beriberi, namely, edema, peripheral neuritis and cardiac failure, is present.

The diagnosis of a moderate degree of thiamin deficiency is usually not difficult, and with increasing recognition of the fact that peripheral neuritis is usually due to thiamin deficiency, the diagnosis is being made much more commonly than it was a few years ago. Recognition of cardiovascular symptoms as being due to thiamin deficiency has been a much more recent development and, because of this fact and the nonspecific character of the symptoms and findings, namely, dyspnea and palpitation on exertion, edema, and circulatory failure, the diagnosis is more difficult to establish.

Thiamin deficiency is usually to be found in chronic alcoholics, among patients who have been on a limited diet as a result of economic disability, faddism, ignorance or in an attempt to reduce weight, and during pregnancy and lactation.

There are no laboratory procedures which are helpful in determining the adequacy of the thiamin metabolism of a patient. Recently, several biologic tests revealing the apparent thiamin content of tissue fluids or of urine have been developed, but they are not of sufficient simplicity and perhaps accuracy to permit their use in the ordinary clinical laboratory. However, there are two other methods which will enable the physician to suspect that symptoms of his patient are due to thiamin deficiency. These are the calculations of Cowgill's formula and the therapeutic test. In cases in which the previous diet is known, an estimation of the adequacy of the thiamin content of the diet, as compared with the requirement of the patient, may be made by Cowgill's formula. Cowgill has shown that the requirement for thiamin is proportional to both intake of calories and body weight.

The therapeutic test with thiamin may be very useful in establishing a diagnosis of previous deficiency, but great care must be exercised in regard to the interpretation of the clearing up of cardiovascular symptoms, because frequently associated methods of treatment such as rest in bed, sedatives, digitalis, and psychic influences, may be the significant curative factors.

Evidence for thiamin deficiency of very mild character has not been established because of lack of chemical or other direct methods of determining the status of thiamin metabolism in patients with clinically recognizable deficiency.

Not infrequently, in association with thiamin deficiency, will be found evidence of deficiency of other components of the vitamin B complex,

TABLE 6.—*Vitamin B Deficiency (Other than in Thiamin)*

Clinical Changes	Deficiency of	Tests
Pellagra	Nicotinic acid Thiamin Other factors?	Therapeutic only
Macrocytic anemia	Uncertain	Therapeutic only
Gastro-intestinal: glossitis, anorexia, achlorhydria, hypotonicity, hypomotility	Uncertain	Therapeutic only

such as gastro-intestinal symptoms or macrocytic anemia. These symptoms and findings may prove helpful in establishing the correct diagnosis.

Treatment.

In the treatment of thiamin deficiency the exhibition of all of the following measures will be found most helpful: an adequate diet high in vitamin B content, preparations of brewers' yeast or wheat germ and crystalline thiamin chlorid. While crystalline preparations are available they should not, for two reasons, be used exclusively in the treatment of thiamin deficiency: first, because, as has been pointed out, most patients with thiamin deficiency have evidence of deficiency of other components of the B complex, and secondly, because crystalline thiamin chlorid is still quite expensive.

For those patients with only moderate deficiency, and without evidence of impairment of gastro-intestinal function, the use of 30 grams of powdered brewers' yeast of good potency, or 6 grams of autolyzed yeast, administered three times daily, is generally adequate. Along with this should be a high caloric, high vitamin diet, if it can be tolerated, and from 10 to 40 milligrams of thiamin chlorid daily, intramuscularly or orally administered. For those patients with severe cardiac symptoms, daily intramuscular or intravenous injections of similar doses of thiamin may be advisable. In addition, dilute liver extracts, suitable for intramuscular injection, given in doses of 10 to 20 cubic centimeters or more daily, are helpful in controlling glossitis and cutaneous manifestations of the type associated with pellagra. As soon as the patient shows evidence of distinct improvement, the dose of crystalline thiamin may perhaps be reduced to 10 to 15 milligrams daily; but it is advisable to maintain large doses of thiamin and other portions of the vitamin B complex, in the form of food and of yeast and wheat germ.

DEFICIENCY OF VITAMIN B (OTHER THAN THIAMIN)

The vitamin B complex consists of a variety of factors other than thiamin, such as riboflavin, the P-P factor (nicotinic acid?), a filtrate factor, vitamin B_6, and others. However, so far only two members of the complex, namely, thiamin and the P-P factor, have been linked unquestionably with deficiency disease in man. (Table 6.)

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(To be continued)

THE ROENTGEN DIAGNOSIS OF DISEASES OF THE ILEOCECAL REGION OF THE GASTRO-INTESTINAL TRACT*

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PART I

INTRODUCTION

THERE are a large number of disease entities which characteristically affect, and show a special predilection for, the ileocecal region of the gastro-intestinal tract. The purpose of this communication is to present a topographical classification of these conditions, in an attempt to facilitate the roentgen diagnosis. The conditions which are noted in the ileocecal segment of the intestine present a varied symptomatology, and differentiation on a clinical basis alone is difficult. In many instances, the diagnosis rests ultimately on the roentgen examination. The differentiation is particularly difficult in those instances where there is a history of pain in the right lower quadrant of the abdomen, associated with a palpable mass. Many of these conditions present a very similar roentgen appearance, and it is only by a consideration of all the possibilities, that we may hope to arrive at the proper diagnosis. In this regard, a topographical classification would seem to be of value.

A CLASSIFICATION OF DISEASES AFFECTING THE ILEOCECAL REGION FROM THE ROENTGENOLOGICAL STANDPOINT

I. Congenital Anomalies.
   (a) Transposition of the colon.
   (b) Anomalies of descent of the cecum.
   (c) Inverted cecum.

II. Ileocecal Bands.
   (a) Congenital.
   (b) Adhesions (postoperative).

III. Benign Tumors.
   (a) Fibroma.
   (b) Adenoma.
   (c) Papilloma.
   (d) Lipoma.
   (e) Angioma.
   (f) Myoma.
   (g) Carcinoid (argentaffine tumors).
   (h) Endometrial implants.
   (i) Mucous cysts.

IV. Malignant Tumors.
   (a) Carcinoma.
   (b) Sarcoma.

V. Specific Granulomas.
   (a) Ileocecal tuberculosis (primary and secondary).
      1. Ulcerative.
      2. Hyperplastic.
   (b) Actinomycosis.
   (c) Amebiasis.
      1. Amebic colitis (localized or diffuse).
      2. Amebic granuloma.

* From the Department of Radiology of the Queen of Angels Hospital, Los Angeles.

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